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# Paradox of Peramorphic Paedomorphosis: Heterochrony and Human Evolution

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*ABSTRACT* This paper reviews Gould's clock model for heterochronic processes and uses that model to develop simple matrix representations of growth and shape change. Matrix representations of growth and development provide a common formulation for all heterochronic processes. In particular, we show how neoteny can be diagnosed using such a matrix approach. The literature is rife with contradictory representations of how neoteny affects growth allometries and the timing of developmental events, and therefore of the role of neoteny in human evolution. Through the use of multivariate models, we explore these relationships and the internal consistency of opposing views.

Gould's neoteny hypothesis for human evolution has been criticized for a number of reasons. Humans do not grow slowly. The slopes of our growth allometries show no common pattern of change vis-à-vis those of our closest relatives. Humans prolong rather than reduce rates of growth and development of body parts; the brain, for example, ceases growing later in humans than in apes, but during this prolonged period of early ontogeny, it grows at a rapid pace.

This paper evaluates Gould's hypothesis and its critiques by focusing on particular questions. Does neoteny imply slow growth? Does it imply a unidirectional change in the rates of growth of traits? Under neoteny, should the brain cease growing in ancestor and descendant at the same age? Does prolongation of phases of growth and development confute neoteny? On the other hand, is paedomorphosis an inevitable consequence of prolonged growth and development? We show that, for all of these questions, the answer is no. © 1996 Wiley-Liss, Inc.

We have evolved our massive brains largely by the evolutionary process of neoteny: the slowing down of developmental rates and the consequent retention to adulthood of traits that mark the juvenile stages of our ancestors. . . . Neoteny . . . slows down our maturation and gives us a long period of flexible childhood learning. I believe that the analogy between childhood wonder and adult creativity is good biology, not metaphore. (Gould, 1987:63.)

. . . human heterochrony has been the locus of a great number of misconceptions. Indeed, it is almost a proxy for all the gibberish generated in the study of heterochrony writ large. . . . That humans are neotenic is one of the most widely circulated bits of misinformation in both the scientific and popular lit-

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erature. (McKinney and McNamara, 1991:xi.)

## THE PARADOX OF PERAMORPHIC PAEDOMORPHOSIS

In *Ontogeny and Phylogeny,* Stephen Jay Gould (1977) provided a template for the study of heterochrony—the evolution of ontogeny. Gould's primary goal was to explore how changes in developmental patterns lead to phylogenetic modifications in adult morphology. He described heterochronic processes and products using a model that featured an ancestral ontogenetic clock on which descendant ontogenetic trajectories could be mapped. Central to Gould's effort was his conviction that fairly simple epigenetic perturbations often underlie complex morphological evolutionary changes.

One goal of Gould's book was to show how one such simple heterochronic trend had dominated human evolution. Reviving an argument put forth by Bolk (1926), Gould applied the concept of neoteny—"dissociation" of "size" and "shape," with "retardation of shape" relative to size and age at sexual maturation—to human evolution. Humans had become increasingly paedomorphic (i.e., with mature adults retaining increasingly juvenile ancestral characteristics) through the "pervasive retardation of development" relative to growth and reproductive maturation. Of course, Gould recognized that humans are not perfect neotenes; adult humans are not reproductively mature, large-bodied, juvenile apes. "No Darwinian supporter of retardation as a major element in human evolution can deny that many distinctive features are not paedomorphic," Gould observed (1977:363). "The concept of mosaic evolution practically requires such a belief." In the category of features molded by forces other than neoteny, Gould placed postcranial adaptations for upright posture, some (but not all) changes in the form of the basicranium, changes in the sequence of dental eruption, and so on. Neoteny, Gould thought, was responsible for the paedomorphic shape of the adult human skull (relatively large cranial vault, small jaws and marked facial orthognathism), as well as numerous changes in our life history schedule.

In describing neoteny, Gould drew upon a rich historical base in developmental biology. Largely following Kollmann (1885), he defined neoteny as "paedomorphosis (retention of formerly juvenile characters by adult descendants) produced by retardation of somatic development" (Gould, 1977:483). To Gould, somatic (or non-germinal) development is one of three fundamental processes of ontogenesis that heterochrony must address; the other two are reproductive maturation (i.e., germinal development) and growth. Neoteny meant retardation of somatic development relative to both growth and reproductive maturation.

Gould's attempt at resurrecting Bolk's retardation hypothesis for human evolution was received with skepticism, particularly within the anthropological community. Bolk's neoteny hypothesis had strongly non-Darwinian and racist overtones. Gould (1977:356) had tried to separate the "baby from bath water"—placing Bolk's neoteny hypothesis squarely on Darwinian and non-racist footings: "I want to rescue Bolk's data—and his basic insight—from the evolutionary theory to which he tied it and from which it has not been adequately extracted." Yet even his version was regarded as simplistic and non-Darwinian. Rather than accepting a suite of sometimes conflicting selective pressures operating through time and space to finally produce an upright, large-brained, and highly aberrant ape, Gould seemed to want one simple heterochronic perturbation to explain most of human evolution. With a few exceptions (e.g., Montagu, 1989; Groves, 1989),[1] most researchers found Gould's notion that humans are "essentially" neotenous untenable (e.g., Løvtrup, 1978; Eccles, 1979; Dean and Wood, 1984; Bromage, 1987; Bogin, 1988; Shea, 1989; McKinney and McNamara, 1991).

During the 1980s, critics sought to put the human neoteny hypothesis to rest once and for all. Gould's own clock model could be used to generate a set of quantitative predictions that could then be tested against the data of human evolution. For example, Gould had suggested a relationship between growth allometry and heterochrony (the evolution of ontogeny). Ancestral and descendant growth allometries could be used to test the neoteny hypothesis.

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[1] But see Groves' (1991) retraction in the most recent printing of this book (p. 322).

Gould had also suggested a relationship between developmental timing and developmental rates. His assertion—that developmental retardation (or the reduction of the rate of change of shape) increases the duration of developmental stages—could be tested against the predictions of his "neoteny" clock. Clearly, humans had undergone a prolongation of life history stages, as exemplified, for example, by the timing of dental eruption. Gould cited such data as proof of an important role for neoteny in human evolution. The question was, Does neoteny require prolongation of life history stages? Does Gould's clock graph depicting neoteny match the data for human evolution?

In a review article published in the *Yearbook of Physical Anthropology,* Shea (1989) concluded emphatically that it does not. According to Shea, Gould had applied neoteny to human evolution without fully realizing the empirical implications of his own model. Not only had Gould applied his model selectively, but even many of his selected applications were wrong. To the small extent that humans are paedomorphic, we probably did not get that way via neoteny.

McKinney and McNamara (1991) were yet more emphatic in their rejection of an important role for neoteny in human evolution. Shea (1989) had admitted the possibility that neoteny had played a role in the evolution of the human brain. McKinney and McNamara vehemently denied that possibility.

Both Shea (1983, 1984, 1988) and McKinney (1986, 1988) developed formalisms for diagnosing neoteny and other heterochronic processes. Shea described neoteny as having two requirements. First, the descendant's growth allometries (the relationships between rates of growth of traits and rates of overall growth in size) must deviate from those of the ancestor. In other words, there must be a "dissociation" of descendant from ancestral growth allometries. Second, the slopes of descendant growth allometries should reveal a reduction in the "rate of growth of shape" relative to the "rate of growth of size."

Exactly how, under a reduction of the "rate of growth of shape," the slopes of descendant growth allometries should differ from those of their ancestors was suggested more by examples than by explicit description. Alberch et al. (1979) had depicted neoteny as a reduction in the rate of change of shape over ontogenetic time. In describing pygmy chimpanzees, Shea (1983, 1984) illustrated neoteny as effecting downward shifts in the slopes of skull/trunk growth allometries. The main thrust of his argument, however, was not that neoteny produces a directional shift in the slopes of growth allometries, but that neoteny, along with acceleration, disrupts the ancestral patterns of covariance in size and shape. To Shea, a fundamental question that heterochrony must address is the coincidence ("association") or non-coincidence ("dissociation") of ancestral and descendant growth allometries. Coincident ancestral and descendant growth allometries, or "ontogenetic scaling," is not an expectation of neoteny or acceleration.[2]

Shortly following Shea's (1983, 1984) discussions of the relationship between allometry and heterochrony, McKinney (1986) produced a set of allometric diagnostics for almost all of the heterochronic perturbations discussed by Alberch et al. (1979) (Fig. 1). McKinney (1986) argued that each heterochronic process could be identified by its effects on the slopes and intercepts of growth allometries; he later qualified this statement by showing how his diagnostics might not work when size is not a simple increasing function of age (McKinney, 1988). During the 1980s, both Shea's and McKinney's formalisms became tools of choice for heterochronic diagnosis. Numerous publications depended on them. Their appeal derived from the ease with which they could be applied, and their apparent ability to distinguish among different heterochronic perturbations—separa-

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[2] In Shea's diagnostic scheme, "ontogenetic scaling" can indicate either a change in the duration of growth (time hypo- or hypermorphosis), or a change in the rate of growth-in-time (rate hypo- or hypermorphosis). Shea's "time hypomorphosis" and "time hypermorphosis" are equivalent to Gould's (1977) and Alberch et al.'s (1979) "progenesis" and "hypermorphosis," *respectively.*

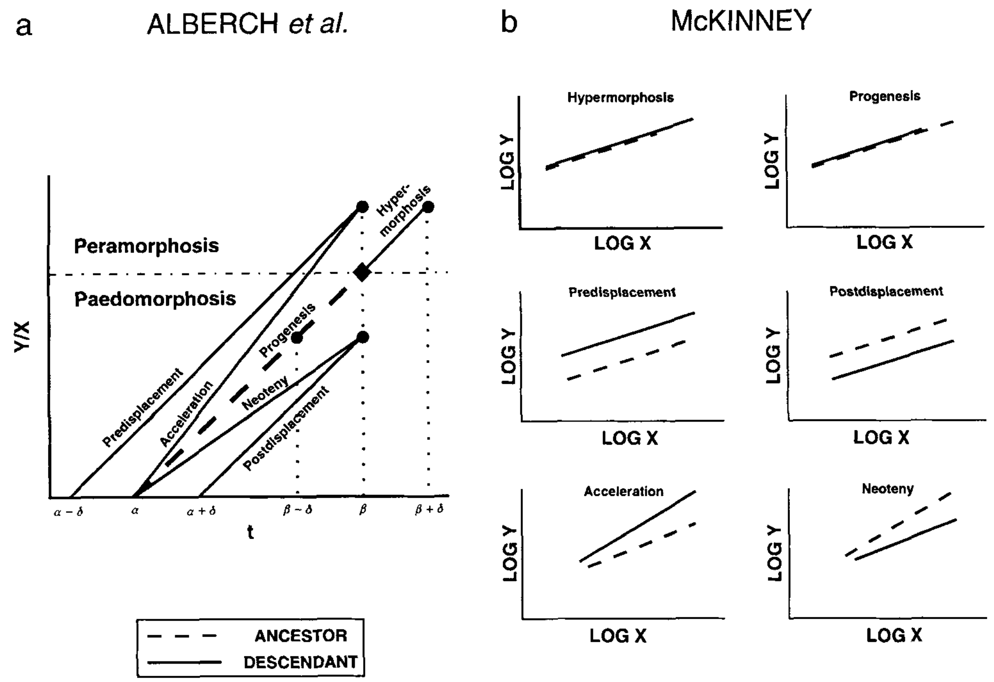

Fig. 1. **a:** Alberch et al.'s (1979) idealized formalism for diagnosing heterochronic processes on bivariate (age-shape) scales. The ratio $Y/X$ represents shape; $t$ represents age. The dashed line represents the ancestral ontogeny and solid lines represent possible descendant trajectories. The solid square represents the ancestral adult, and solid circles represent possible descendant adults. Designating the slope as the "rate of growth" of shape, $\alpha$ as the age of "onset" of shape, and $\beta$ as the age of "offset" of shape, heterochronic perturbations can be diagrammed as changes ($\delta$) in each of the three parameters. Thus "rate change" (acceleration or neoteny) is measured by a change in the slope of the "shape growth" trajectory, and changes in offsets and onsets are represented by perturbations in $\beta$ and $\alpha$, respectively. Peramorphosis occurs when descendant adult shape transcends that of the ancestor; paedomorphosis occurs when descendant adult shape matches that of an ancestral juvenile. (Modified from Klingenberg and Spence, 1993; based on Alberch et al., 1979.) **b:** McKinney's (1986, 1988) formalism for diagnosing heterochronic processes. The six processes presented by Alberch et al. (1979) are transplanted onto logarithmically transformed size-trait scales. Heterochronic processes are inferred from changes in the intercepts and slopes of growth allometries. Neoteny is diagnosed as a decrease in the value of the slope, $k$. (Modified from McKinney and McNamara, 1991.)

ting those induced by shifts in timing from those generated by shifts in rate.

Gould's retardation hypothesis for human evolution was tested against this quantitative framework and found to be wanting. Critics maintained:

1. Neoteny entails a reduction in the rate of growth of shape. In no sense do humans grow slowly; therefore, we are not neotenic (McKinney and McNamara, 1991).

2. Humans did not evolve through a single heterochronic perturbation. Human growth allometries are perturbed (cf. those of apes) in diverse directions. Dean and Wood (1984) observed an internal inconsistency in the fact that Gould attributed to neoteny both increases and decreases in the rates of growth for different traits.

3. Gould (1977) had conflated slow shape change with prolongation of growth and delayed maturation (see especially Shea, 1989; McKinney and McNamara, 1991). Neoteny is about developmental rates, not timing; prolongation of growth entails a change in timing. Rather than showing a reduction in their rate of growth of shape (cf., size), humans exhibit delayed onsets and "offsets"[3]

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[3] The literature on heterochrony uses the term "offset" as an antonym for onset—i.e., to mean cessation; see Alberch et al. (1979) and subsequent literature.

of particular events in developmental sequences.

This last criticism, in particular, has been repeated by many detractors of Gould's hypothesis. In a review of *Ontogeny and Phylogeny,* Løvtrup (1978) suggested that prolongation of fetal growth characteristics—not retardation—describes evolutionary changes in human development. Bogin (1988) saw the insertion of a whole new phase of development (childhood) between infancy and juvenescence as contrary to the expectations of neoteny. Shea (1989: 80) was most explicit: Neoteny predicts no change in the timing of developmental events—only a reduction in the rate of change of shape. McKinney and McNamara (1991) took Shea's argument one step further: prolongation of phases of growth or development leads not to paedomorphosis (retention of juvenile ancestral features in mature descendants) but to peramorphosis (overdevelopment of ancestral adult features in mature descendants). A delayed cessation of brain growth, and the resultant increase in neurocranial size, is a prediction of hypermorphosis (late "offset" timing), and not of retardation in the rate of growth of shape.

To make the quantitative predictions of neoteny more explicit, Shea (1983, 1984, 1989) offered an example of an actual case of neoteny within the Hominoidea (Fig. 2). Adult pygmy chimpanzees have small, juvenilized skulls, long limbs, and long trunks. Assuming an ancestor for pygmy chimpanzees similar to *Pan troglodytes,* Shea claimed that this combination of features had evolved via a neotenic reduction in the rate of change in skull shape relative to overall growth. He defended this example as conforming closely to the predictions of Gould's model for "pure neoteny." Support for this claim was derived from the following observations: 1) there is no significant difference between pygmy and common chimpanzees in age at maturation; 2) there is no significant difference in "overall" growth (as reflected by the size of the limbs and trunk); and 3) the slope of the ontogenetic allometry of skull to trunk length is lower in pygmy chimpanzees than in common chimpanzees.

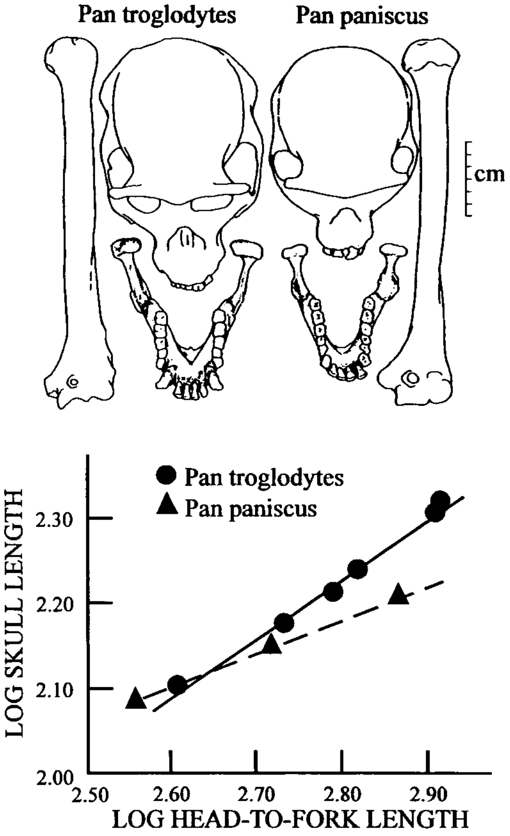

Fig. 2. **Top:** Relative sizes and shapes of the adult skull and humerus of two species of *Pan.* **Bottom:** Shea (1983) diagnoses "true neoteny" in pygmy chimpanzees (cf. common chimpanzees) based on the lower slope of their skull/trunk growth allometry. (After McHenry and Corrucinni, 1981; Shea, 1983.)

In this paper, we develop a framework for evaluating hypotheses derived from Gould's clock graphs. We believe that Gould's clocks are unambiguous and completely testable. They can be used to derive predicted changes, under particular heterochronic perturbations, in the slopes and intercepts of growth allometries. It is also our belief that many of the criticisms that have been leveled against Gould's hypothesis for human evolution are misleading or wrong. They appear to be grounded in misinterpretations of how "retardation" in the rate of change in shape (sensu Gould, 1977; or Alberch et al., 1979) would affect 1) growth allometries; and 2) the timing of developmental events. As a result of these misconceptions, critics have inconsistently and sometimes incorrectly described what neoteny means and how it can be tested.

Our task in this paper is fivefold:

1. First, we reexamine neoteny through Gould's perspective. We make explicit Gould's definition of neoteny, and what the "size," "shape," and "age" vectors on his heterochronic clock represent. What does dissociation of "size" and "shape," or dissociation of "shape" and "age," actually mean?
2. We show how any clock graph can be represented as multivariate ancestral and descendant age-size-shape matrices that can be used to derive a set of specific predictions regarding changes in growth allometries and the timing of developmental events.
3. We review the contradictory ways in which allometry has been used to draw inferences regarding heterochrony. We show how McKinney's (1986, 1988) popular diagnostic formalism does not address the dissociation of growth and development which is so critical to neoteny as described by Gould (1977).
4. We examine the relationship between developmental rates and developmental timing. What does a reduction in the rate of change in shape mean, and how does this relate to the tempo of development? Is Gould correct in assuming a relationship between a "matrix of retardation" (1977:376) and paedomorphosis?
5. We offer suggestions for further testing Gould's hypothesis that humans are "essentially" neotenic.

## GOULD'S "NEOTENY"

If Gould's hypothesis is to be fairly tested, the starting point of reference must be Gould's definition of neoteny. Gould (1977, p. 228) used "neoteny" in a manner consistent with Kollmann's (1885) original definition. Without any change in the age of reproductive maturation, a reduction in the rate of somatic development (or rate of change in form) can produce paedomorphosis. This is neoteny: paedomorphosis through slow development.

So defined, neoteny can be distinguished from other means of generating paedomorphosis. In all cases, paedomorphosis requires a decrease in net development. Shape at maturation must become "juvenilized." Specific processes of heterochrony (e.g., progenesis, neoteny) show how such juvenilization can occur. For example, it can be generated by accelerating sexual maturation, without changing the rates of growth and development. The result is a descendant whose growth and development have both been eclipsed. This, however, is progenesis; it is not neoteny as described by Kollmann (1885).

Reduced rates of somatic development need not carry with them any modification in the net rate of growth. If development slows without effecting any other change (i.e., either a decrease in net growth or a delay in the age at maturation), then the mature descendant will be "juvenile" in shape but not in size. Such a descendant—a "pure neotene"—will match its ancestor in size and age at maturation, but not in adult shape.

Gould (1977) found intriguing the observation that, within evolutionary lineages, the three fundamental processes of ontogenesis (growth, development, and maturation) are potentially dissociable. Whereas a perturbation in one process normally affects the others, each is also capable of evolving independently. A slowly developing and late-maturing species need not also grow slowly. Gould developed his celebrated clock model precisely to assess and to display independent (dissociated) as well as linked (associated) changes in growth, development, and maturation.

Gould was explicit in the meanings he attached to these terms. Growth meant ontogenetic change in "size" ($\Delta X$). Size has length, area, volume, or mass; size variables must have mensurable units (e.g., millimeters, grams, cubic centimeters, etc.).

Development meant ontogenetic change in body "shape"—the form of somatic (or nongerminal) structures. Shapes are ratios ($Y/X$) or angles ($A$) describing proportions or relative positions of structures. Development is change in shape—$\Delta[Y/X]$ or $\Delta A$. Because shape is dimensionless, there is no requirement that shape values increase over time. Specific proportions or angles can increase, decrease, or stay the same. For ratios, this depends on whether trait size ($Y$) increases with positive allometry, negative allometry, or isometry with respect to overall size ($X$). Ratios or angles can even reverse direction with age.[4]

Maturation meant the ticking of biological "time"—i.e., the ontogenesis of the reproductive apparatus. It can be measured as the net change in age ($\Delta t$) from $t_i$ to $t_m$ (initiation of development to some selected maturational stage—generally sexual maturation or adulthood).

Association or dissociation of growth, development, and maturation meant the maintenance (=association) or disruption (=dissociation), in the descendant, of ancestral relationships between $\Delta X$, $\Delta[Y/X]$, and $\Delta t$ (from $t_i$ to $t_m$).

The task of the clock was to provide a pictorial comparison of ancestral and descendant growth, development and age at a common stage of maturation. Clocks are, in effect, unconventional means of comparing ancestral and descendant matrices. They require that a descendant's ontogenetic paths be plotted on scales that are calibrated according to ancestral trajectories for growth, development, and maturation. From a specific ancestor, a particular clock indicates how a given descendant might arise.

## CONSTRUCTING AND CALIBRATING CLOCKS

Envision, first, an ancestral matrix comprising three column vectors: age ($t_{\mathrm{ANC}}$), shape ($[Y/X]_{\mathrm{ANC}}$), and size ($X_{\mathrm{ANC}}$) (Fig. 3a). The values in the cells represent species norms. Age is recorded at equal intervals from $t_{i\,\mathrm{ANC}}$ to $t_{m\,\mathrm{ANC}}$, and the corresponding values for shape and size are registered. This process fixes (or naturally "calibrates") the scales of shape and size.

Age is a linear vector. Size and shape are only occasionally linear with respect to each other, and virtually never with respect to age. Because age is linear, equal distances along the age vector represent equal amounts of time. If an ancestor is 6 years old at maturation ($t_{m\,\mathrm{ANC}} = 6$), then it is 2 years old when it has reached one-third of the distance along the age vector. Because shape is not a linear vector, one cannot infer the value of shape at any point along the shape pathway without knowing how shape scales. The fact that a 2-year-old ancestor has progressed one-third of the way along its shape pathway provides no information whatsoever about the absolute amount of shape change that it has experienced to that point. Ditto for size. To calculate the exact amounts of change incurred during any period of time, one must know the values of size and shape in the cells of that sector of the matrix. Knowing the scales of shape and size also allows one to calculate rates of change. Rates can be measured at any instant of time or over any period of time.

To set the scales for a clock, Gould simply turned the ancestor's three-column matrix counterclockwise onto its side, separated the shape and size vectors from the age vector, and molded the shape and size vectors into arcs of different lengths to fit, one inside the other, along the periphery of half of a semicircular clock (Fig. 4). Although the absolute lengths of the vectors have changed, no transformation or linearization of shape or size has occurred.[5] The calibrated clock is simply a pictorial representation of three vectors, one of which lies horizontally along the baseline, and two of which lie along the peripheral arcs. Corresponding vector cells for size, shape, and age can be located by converting proportional distances into radians or degrees. For example, the values for size and shape at $t_{i\,\mathrm{ANC}}$ are located near 0°; the values at $t_{m\,\mathrm{ANC}}$ are located at the clock's midline (90°); and the values of size and shape at $t_m/3$ are located at the 30° mark.[6] The scales of shape and size are still exactly

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[4] Net change in shape can be conceived as an absolute value—$\Delta|Y/X|$ or $\Delta|A|$. So conceived, change in shape is positive whether or not the value of the ratio or angle increases. This is implicit in Gould's treatment of particular exmples. When the value of the ratio decreases with time, the ratio is plotted on reverse scales (large to small values) such that the acquisition of increasingly small ratio values is visually interpreted as an increase in net development.

[5] When, as in paleontological examples, age is not known, Gould calibrated shape directly against size (and not age). He might then linearize size, and plot size and shape along the clock's periphery, but with the caveat that neither the relationship of size nor that of shape to age could be read directly off the block.

[6] Ontogeny begins at conception, but shapes do not develop until later, and they cannot be measured until yet later. Thus we set $t_i$ near, rather than at, 0°.

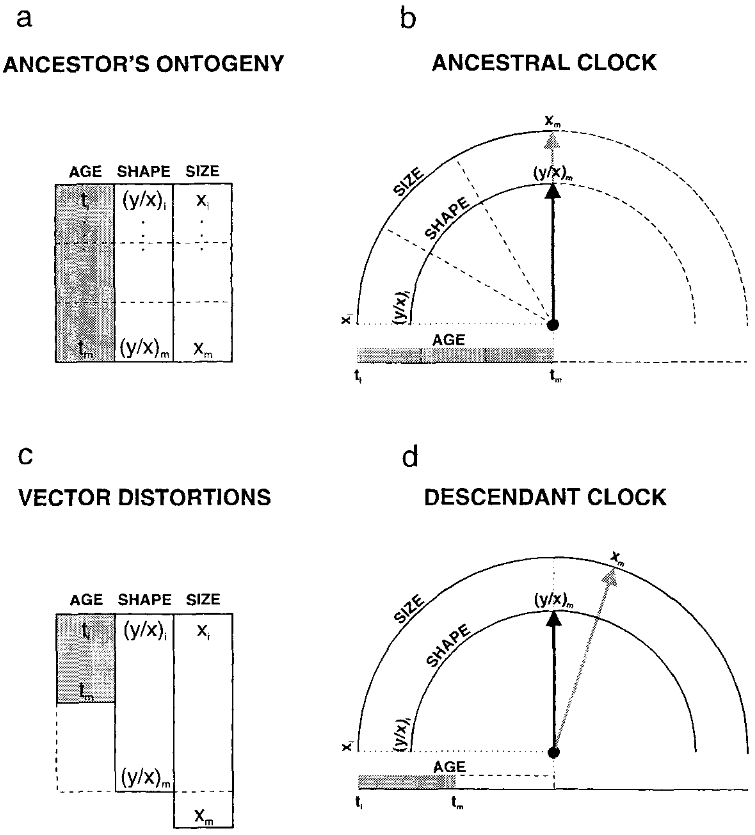

Fig. 3. **a:** The clock as a comparison of ancestral and descendant sets of triples: i.e., age (or $t$), shape, and size, all indexed on $t$, from the age of initiation of development ($t_i$) to maturation ($t_m$). **b:** The ancestral set of triples is used to calibrate the clock. Corresponding row cell values can be read off the clock by converting proportional distances to radians. Dashed lines show the placements of corresponding cells (here, values for size and shape at $.33t_m$ and $.67t_m$) in the matrix and on the calibrated clock. The descendant set of triples is used to diagnose heterochronic process. By mapping descendant age, shape, and size at maturation onto scales that are calibrated according to ancestral trajectories, the distortions of the ancestor needed to produce a particular descendant become clear. These distortions can be represented in matrix form **(c)**, or as a clock diagram **(d)**.

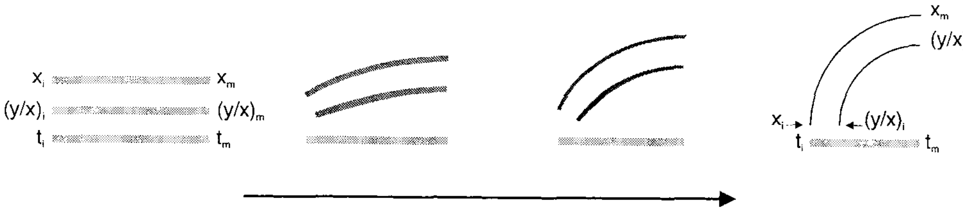

Fig. 4. Pictorial conversion of an ancestral age-shape-size matrix into a clock diagram. See text.

as they were in the initial rectangular matrix (Fig. 3b).

Having set the scales of the clock according to ancestral paths of growth, development, and maturation, the clock is now ready to be used as a comparative tool. Descendants can be represented as vectorial distortions of ancestral clock vectors, as long as one is willing to constrain descendants to follow ancestral shape pathways. The descendant is "mapped" onto the ancestor's clock by marking its size, shape and age at maturation on the ancestor's clock. If, at the selected maturational stage, the descendant has attained a larger size in half the time without changing shape, the size vector will have stretched, the shape vector remained the same, and the age vector will have shrunk (via truncation) (Fig. 3c,d).

A descendant's clock shows how the ancestor's clock must be distorted to produce that particular idealized descendant. The idea is similar in principle to D'Arcy Thompson's (1917) deformed-coordinate representations of changes, from ancestor to descendant, in geometric shape (Fig. 5a). The deformed coordinates, by themselves, show the amounts and directions of deformation needed to produce a given descendant (Fig. 5b). Actual measurements of the ancestor on the original rectangular grid are needed to reconstruct the shape of the descendant.

In the example shown in Figure 3c and d, the descendant's vector for age is half the length of that of its ancestor. Its shape vector is equal in radians (or arc length) to that of its ancestor. The length of the size vector has increased by 20%. On the clock, this is registered by a change in the position of its terminus, from 90° to 108° [i.e., 90° + .2(90°) = 108°].

Because the scales for size, shape, and age are known, one can translate these vectorial distortions into amounts and rates of change of size and shape in the descendant. Age is linear; thus, at 50% of the vector's length, $t_{\mathrm{m\,DES}}$ is exactly half the value of $t_{\mathrm{m\,ANC}}$ ($t_{\mathrm{m\,DES}} = .5\ t_{\mathrm{m\,ANC}}$). Shape is not linear, but, in this example, the descendant's shape at maturation ($[Y/X]_{\mathrm{m\,DES}}$) is easy to determine, because it is equal to that of the ancestor. When descendant shape does not match that of the ancestor, its value at maturation must be read off the calibrated clock.

Size is not linear. Thus, as for shape, vector length per se does not reveal the amount of size increase. The exact amount of change in size is given by the scale for size that was determined through the initial clock calibration. Always, in the case of sizes or shapes "beyond" those of the ancestor at maturation, the scales for size and shape are set through extrapolation of the ancestor's paths of change in size and shape.

It should be noted that preservation of ancestral shape at maturation does not imply preservation of ancestral trait size ($Y$). It means only that $[Y/X]_{\mathrm{m\,ANC}}$ has remained unchanged—i.e., $[Y/X]_{\mathrm{m\,DES}} = [Y/X]_{\mathrm{m\,ANC}}$. If X has increased while $Y/X$ remains unchanged, then the size of trait $Y$ must have increased.

Similarly, preservation of ancestral shape at maturation does not imply that the ancestor's rate of development has remained unchanged. The descendant's rate of development is not revealed by its shape at maturation, but rather by the ratio of $\Delta[Y/X]_{\mathrm{DES}}$ to $\Delta t$ (from $t_{\mathrm{i\,DES}}$ to $t_{\mathrm{m\,DES}}$). If the descendant undergoes the same amount of change in shape in only half the time, its rate of change in shape must increase (Fig. 6).

Figure 7 shows a sample clock after calibration. Once the descendant values for growth, development and age at maturation are plotted on such a calibrated clock, the clock can provide an enormous amount of comparative information. One can read ancestral values for age, shape, and size throughout ontogenesis and at maturation. One can read descendant values for age, shape, and size at maturation. One can use the descendant's values for overall size at maturation ($X_{\mathrm{m\,DES}}$) and shape ($Y/X$) at any age ($t$) to determine the descendant's trait size ($Y$) at any age (given the assumption that the descendant has followed its ancestor's shape pathway throughout ontogeny).[7]

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[7] An ancestor's matrix is a set of triples (age, shape, and size). The pairing of shape and size defines their association or linkage in the ancestor. If ancestral linkages between shape and size are broken, then the descendant must have at least one size/shape pair that matches its ancestor in one dimension but not the other. Thus, if the descendant exhibiting size/shape dissociation has the same shape path as its ancestor, it must have a size path that differs from that of its ancestor, and vice versa. Under dissociation of size and shape, a stable shape pathway necessitates a new and different size pathway. In constructing our models, we demanded descendant conformity to the ancestor's shape (rather than size) pathways. We allowed the relationship between shape and size, given their values at maturation, to define the descendant's new size pathway. We did so because we take conformity of the descendant to the ancestor's shape pathways as implicit in Gould's clock model. Unless the descendant follows its ancestor's shape pathways, there is no reason to expect changes in rates of development to yield paedomorphosis or peramorphosis in the adult; they will simply yield something new. In actuality, descendants are not constrained to follow ancestral shape pathways at all. The heterochronic implications of this fact are discussed elsewhere (Sutherland and Godfrey, in prep.).

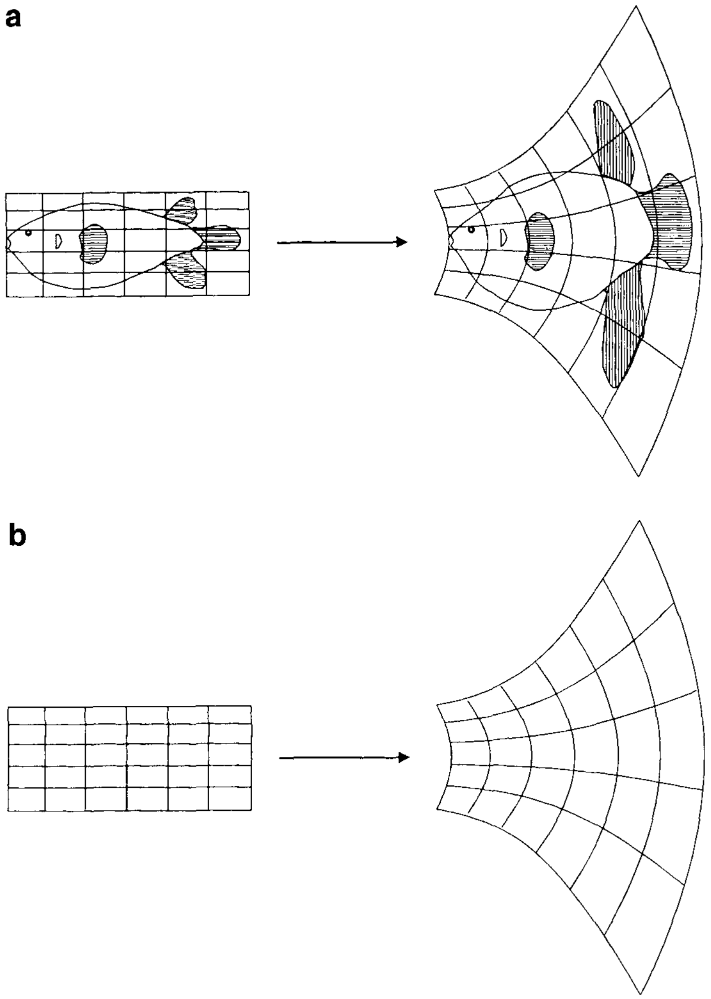


Fig. 5. **a:** D'Arcy Thompson's grid coordinate deformation. **b:** The deformed grid coordinates (like the deformed descendant vectors on Gould's clocks), do not directly describe the descendant; rather they show how the ancestor must be deformed to produce the given descendant.

For example, Figure 7a compares an ancestor that matures at age 6 with a descendant that matures at age 3. The mature ancestor exhibits an overall size, $X_{\mathrm{ANC}}$, of 42 cm and a trait size, $Y_{\mathrm{ANC}}$, of 14.7 cm, or 35% of 42. The mature descendant is 46 cm in size and exhibits a trait size of 16.1 cm (35% of 46). $Y_{\mathrm{ANC}}$ has increased from 1.7 cm (87% of 2 cm) to 14.7 cm, while $Y_{\mathrm{DES}}$ has increased from 1.7 cm to 16.1 cm. The descendant follows its ancestor's shape pathway, but matures in a shorter period of time (3 years) and grows to a larger size (46 rather than 42 cm).

In order to determine corresponding descendant values for age, size, and shape throughout ontogenesis, the descendant's clock must be recalibrated (through interpolation or extrapolation, as required). Figure 7 demonstrates that process: Figure 7a and c show the descendant as a distortion of its particular ancestor, whereas Figure 7b and d show descendant age, size, and shape vectors after recalibration. Figure 7d represents

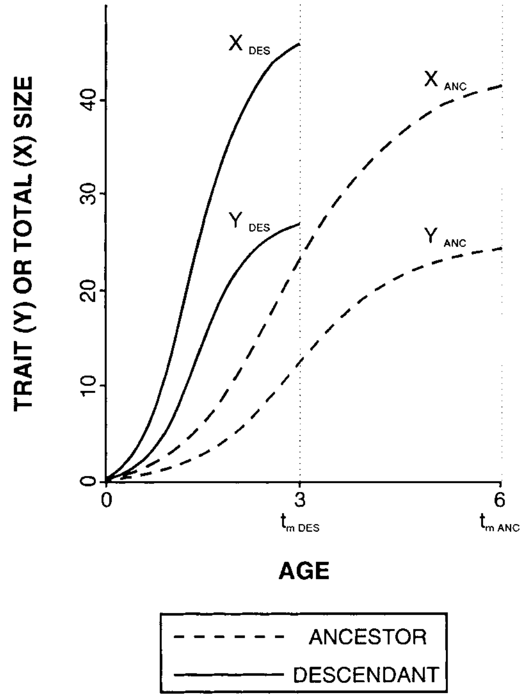


Fig. 6. Comparison of ancestral and descendant growth trajectories. The descendant exhibits an increase in size and maintenance of shape in half the maturational age. The descendant is a rapidly maturing proportioned giant. These growth trajectories match the ancestor and descendant represented on Figure 4.

the descendant's own age-shape-size matrix, with the corresponding descendant values, at each age, of shape and size.

## THE CLOCK AS A METRIC FOR HETEROCHRONIC DISSOCIATION

The advantage of depicting the descendant as a distorted ancestor is that it allows the investigator to infer heterochronic process at a glance. Those processes involve associations and/or dissociations of growth, development, and reproductive maturation. Association is indicated by the identity, in the descendant, of distances and radians traveled along ancestral age, size, and shape vectors. Under association of growth and development (Fig. 8a), the relationship between size and shape in the descendant is exactly as in the ancestor. The descendant's size and shape "hands" are depicted as moving together throughout ontogeny, following ancestral pathways for growth and development, and necessarily matching some ancestral combination at maturation. (This can occur with or without a change in the rates of growth and development.) Association of growth and maturation (Fig. 8b), or of development and maturation (Fig. 8c), is represented by an equivalence of radians and distance traveled along the size and age (or shape and age) vectors respectively. Such association implies that the net rate of growth (or development) in the descendant is identical to that of the ancestor.

Dissociation is indicated by the non-identity, in the descendant, of distances and radians traveled along ancestral age, size, and shape vectors. The descendant represented in Figures 3, 6, and 7, for example, has undergone dissociation of all processes of ontogenesis. It has achieved its ancestor's mature shape in half the time; thus, development and maturation have been dissociated. It has also grown larger than its ancestor in half the time; thus, growth and development have been dissociated. Development has slowed relative to growth, because the descendant does not attain its ancestor's shape until it has reached a larger size. In effect, the rates of growth and development have both increased—growth more so than development—resulting in a net retardation of development relative to growth. The descendant in this example is a rapidly-maturing, "proportioned giant." It is larger at maturation than its ancestor, but it is exactly the same shape (Fig. 6).

Paedomorphosis or peramorphosis can be produced by any of a number of size/shape/age associations or dissociations. The clock graphs depicted in Figure 9 show three means of producing paedomorphosis—progenesis (truncation of growth and development via early maturation), rate hypomorphosis (retardation of both growth and development relative to maturation), and neoteny (retardation of development relative to both growth and maturation).

Each of these clocks can be represented as a set of multivariate age-size-shape matrices. Using multivariate matrix representations, the effects of particular heterochronic perturbations on whole organisms or on in-

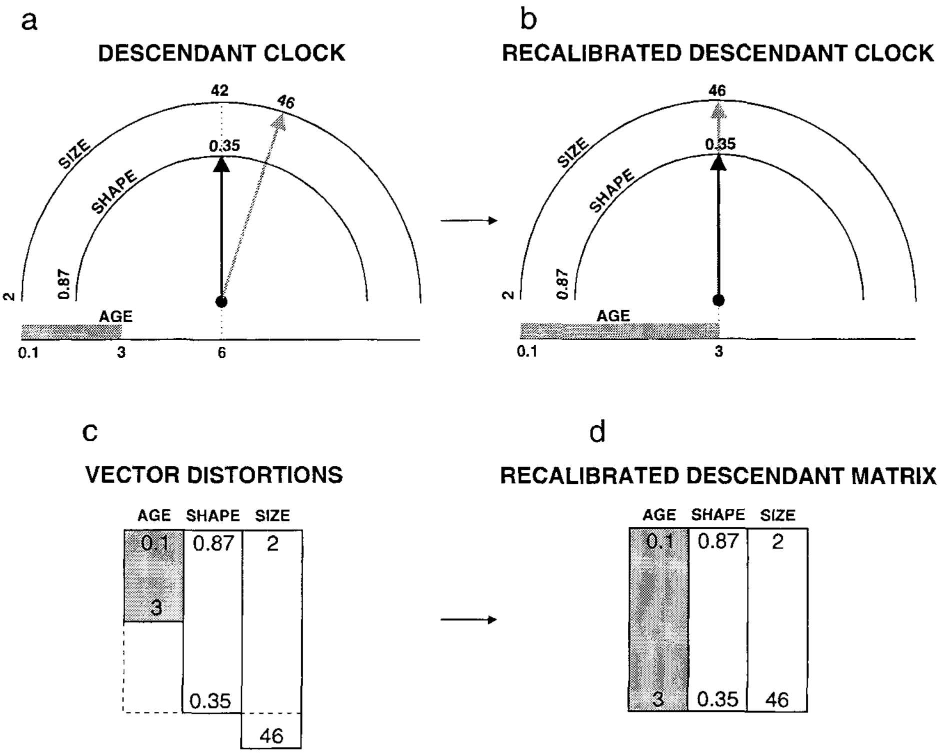


Fig. 7. The same descendant is here displayed as a set of vectorial distortions of an ancestor with known shape and size scales (**a** or **c**). In order to reconstruct the descendant's own values, at each age, of size and shape, size and shape must be recalibrated (by extrapolation or interpolation, as needed) against the descendant's age. The recalibrated descendant can then be represented as a new clock (**b**), or as an undistorted matrix (**d**).

dividual growth fields can be modeled. We turn our attention to this problem in the next section, where we explore the relationship between developmental retardation and changes in the slopes and intercepts of growth allometries.

## HETEROCHRONY AND ALLOMETRY

Gould has stated that neoteny effects an increase in the rates of growth of certain traits and a decrease in the rates of growth of others. Whether a particular trait exhibits an increase or decrease in its relative (or absolute) rate of growth depends on its ancestral growth allometry. Others have asserted, contra Gould, that slowing the rate of development uniformly lowers the slopes of growth allometries. This particular assumption is built into the diagnostic formalism of "allometric heterochrony" constructed by McKinney (1986, 1988; see also McKinney and McNamara, 1991; Wei, 1994) as well as some of the examples of "neoteny" shown by Shea (e.g., 1988, p. 244; 1989, p. 91; but see Shea, 1989, pp. 73, 96).

If Gould is correct, one should be able to model a perfect neotene by slowing down the rate of change in all proportions while *maintaining overall size at maturation*. The descendant should be paedomorphic as an adult; it should show a delay in the timing of all somatic developmental events embedded in the ancestral developmental sequence. Intervals between onsets and offsets of developmental phases should be prolonged. The evolutionary lineage should show a mosaic of changes in rates of absolute and relative growth. Under neoteny, traits that grow with negative postnatal allometry—i.e., that have relatively high fetal (and/

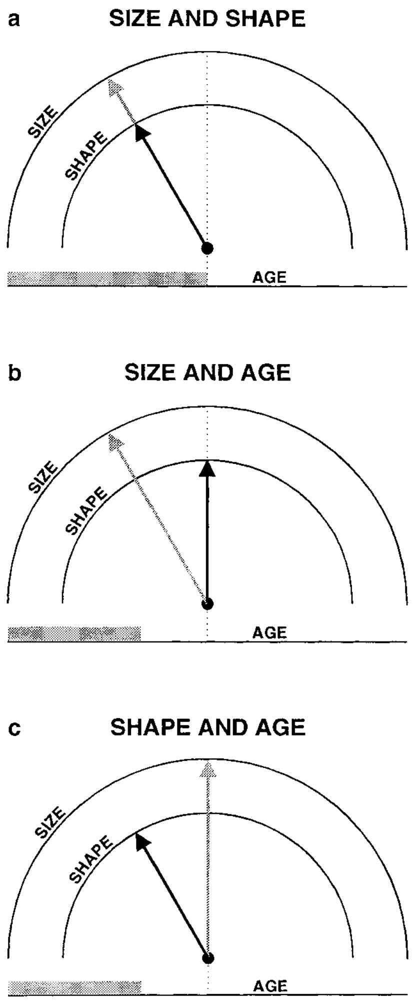


Fig. 8. Retention of ancestral linkages between growth, development, and maturation is represented by a match (in proportional length or radians) of two or more descendant vectors. **a:** Association of growth and development. **b:** Association of growth and maturation. **c:** Association of development and maturation.

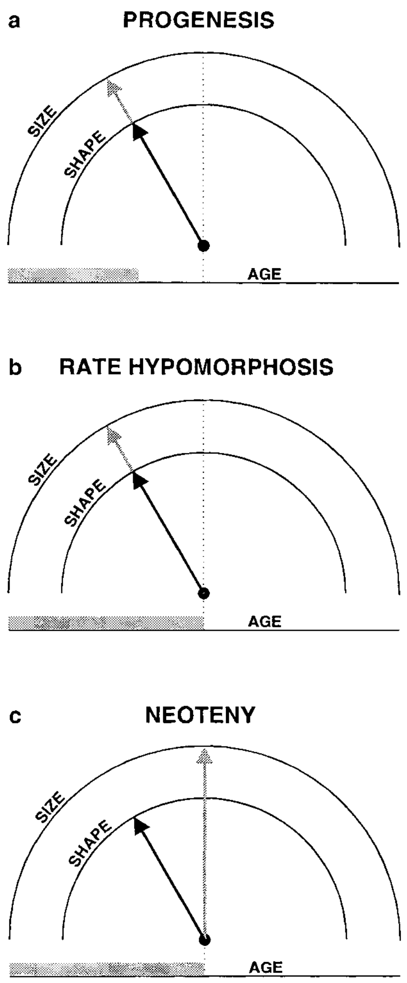


Fig. 9. Three heterochronic processes that result in paedomorphosis. **a:** Progenesis (paedomorphosis through early maturation). Ancestral linkages between growth and development are retained, as are ancestral rates of both growth and development. **b:** Rate hypomorphosis (paedomorphosis without disruption of the ancestral linkages between growth and development, but with retardation of the rates of growth and development). **c:** Pure neoteny (paedomorphosis via retardation of development, but without a reduction in net growth or a change in age at maturation).

or early postnatal) as well as relatively low late postnatal growth rates—should show an increase in their rates of growth relative to size. Traits that grow with positive postnatal allometry should show the opposite.

If, however, McKinney and McNamara (1991) are correct, one should be able to model a perfect neotene by lowering the slopes of all growth allometries while maintaining overall ancestral size at maturation. The descendant should be paedomorphic as an adult, but should show no change in the timing of somatic developmental events embedded in the ancestral developmental sequence. All traits should be uniformly affected, whether or not they grow with negative (or positive) postnatal allometry.

It is a simple matter to model the evolution of a "perfect paedomorph" in multivariate space. A perfect paedomorph is a descendant all of whose adult proportions are identical to those of its ancestor at a juvenile stage. Figures 10 and 11 show two ways to produce a perfect paedomorph from the same hypothetical ancestor. Figure 10 models paedomorphosis via size/shape association; Figure 11 models paedomorphosis via size/shape dissociation—i.e., neoteny.

Under size/shape association, the descendant adult will equal the size of the juvenile ancestor whose shape it mimics. Such a descendant can theoretically evolve through 1) progenesis (=time hypomorphosis, whereby the descendant grows and develops at ancestral rates but growth and development are eclipsed by early maturation); or 2) rate hypomorphosis (whereby the descendant grows and develops at a slower pace, but matures when still at an ancestral juvenile developmental stage). Figure 10 shows paedomorphosis of an entire growth field via rate hypomorphosis. A single clock portrays every age-size-shape comparison.

The first (top) array of vectors and matrices on Figure 10 displays the hypothetical ancestor. The middle array shows distortions necessary to produce a paedomorphic descendant via rate hypomorphosis. The last array shows the descendant's recalibrated trait, size and shape vectors.

At the extreme left of each array is the age vector $(t)$—Gould's measure of maturation. This is a linear vector that begins at the onset of development and ends at maturation; in this case, both ancestor and descendant mature at age $n$. Ancestral values for trait sizes, overall size, and proportional shape are shown for ages $n$ and $p$, where $p$ represents the ancestor's age at some juvenile stage of growth and development.

To the right of each age vector is what might be called the trait matrix. Each column in this matrix represents one metric trait $(Y_i)$. The whole matrix comprises the set of metric traits under consideration: $Y_1$, $Y_2$, $Y_3 \ldots Y_k$. Any number of traits can be considered in a multivariate extension of a clock. We have chosen to base our display on three traits—one that grows in the ancestor with positive postnatal allometry $(Y_1)$, one that grows with negative postnatal allometry $(Y_2)$, and one that grows with virtual postnatal isometry $(Y_3)$ relative to overall size. Whereas trait sizes are never directly represented on Gould's clocks, they are critical to heterochronic analysis because they are an integral part of shape (see below).

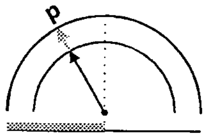
To the right of each trait matrix is the size vector—Gould's measure of growth. Following Gould (1977) and Alberch et al. (1979), we represent overall size as the sum of the trait sizes under consideration. This is not only convenient for multivariate analysis, but it captures the spirit of the size vector in Gould's clock model (the shapes of traits are their proportional relationships, and size is their sum). At any age $(t)$, overall size, $X(t)$, equals $Y_1(t) + Y_2(t) + \ldots + Y_k(t)$. Other definitions of size are possible and are discussed elsewhere (Sutherland and Godfrey, in prep.).

Next is the shape matrix—Gould's measure of development. Each trait vector (or column in the trait matrix) has a corresponding shape vector (or column in the shape matrix). Shape vectors convert trait sizes to proportions of overall size, from $t = t_i$ to $t_m$. At any age, the sum of all trait proportions is 1.

A comparison of ancestral and recalibrated descendant trait/size relationships shows how particular heterochronic perturbations affect growth allometries. Growth allometries measure the relationship between trait growth $(Y_1, Y_2$, etc.) and growth in overall size $(X)$. They are generally depicted on logarithmic scales. As expected, under "pure" rate hypomorphosis, all descendant growth allometries must match those of the ancestor. Trait/size relationships are identical in ancestor and descendant; the only difference is that the descendant's trait/size relationships are eclipsed at a juvenile ancestral developmental stage.

Figure 11 shows how pure neoteny (sensu Gould, 1977) would perturb the same hypothetical ancestor. Because neoteny entails size/shape dissociation, the mature descendant cannot match the size of the juvenile ancestor whose shape it mimics. Net growth (or overall size at maturation) in the descendant will equal that of the ancestor, and the age at maturation will remain unchanged. Development will have slowed relative to both growth and maturation. As in Figure

# RATE HYPOMORPHOSIS



$t_{m\,DES} = t_{m\,ANC}$

$X_{m\,DES} = X_{p\,ANC}$

$[Y/X]_{m\,DES} = [Y/X]_{p\,ANC}$

## ANCESTRAL VECTORS AND MATRICES

| AGE (mo)<br>t | TRAIT (cm)<br>$Y_1$ | $Y_2$ | $Y_3$ | SIZE (cm)<br>$X = Y_1 + Y_2 + Y_3$ | SHAPE<br>$Y_1/X$ | $Y_2/X$ | $Y_3/X$ | $\Sigma[Y_i/X]$ |
|---|---|---|---|---|---|---|---|---|
| 1 | | | | | | | | |
| 2 | | | | | | | | |
| • | | | | | | | | |
| • | | | | | | | | |
| p | 22.5 | 12.6 | 6.3 | 41.4 | 0.54 | 0.31 | 0.15 | 1 |
| n | 44.9 | 20.0 | 11.0 | 75.9 | 0.59 | 0.26 | 0.15 | 1 |

## DESCENDANT AS DISTORTED ANCESTOR

| t | $Y_1$ | $Y_2$ | $Y_3$ | X | $Y_1/X$ | $Y_2/X$ | $Y_3/X$ | $\Sigma[Y_i/X]$ |
|---|---|---|---|---|---|---|---|---|
| 1 | | | | | | | | |
| 2 | | | | | | | | |
| • | | | | | | | | |
| • | | | | | | | | |
| | 22.5 | 12.6 | 6.3 | 41.4 | 0.54 | 0.31 | 0.15 | 1 |
| n | | | | | | | | |

## RECALIBRATED DESCENDANT VECTORS AND MATRICES

| t | $Y_1$ | $Y_2$ | $Y_3$ | X | $Y_1/X$ | $Y_2/X$ | $Y_3/X$ | $\Sigma[Y_i/X]$ |
|---|---|---|---|---|---|---|---|---|
| 1 | | | | | | | | |
| 2 | | | | | | | | |
| • | | | | | | | | |
| • | | | | | | | | |
| n | 22.5 | 12.6 | 6.3 | 41.4 | 0.54 | 0.31 | 0.15 | 1 |

Fig. 10. Rate hypomorphosis in multivariate perspective. See text.

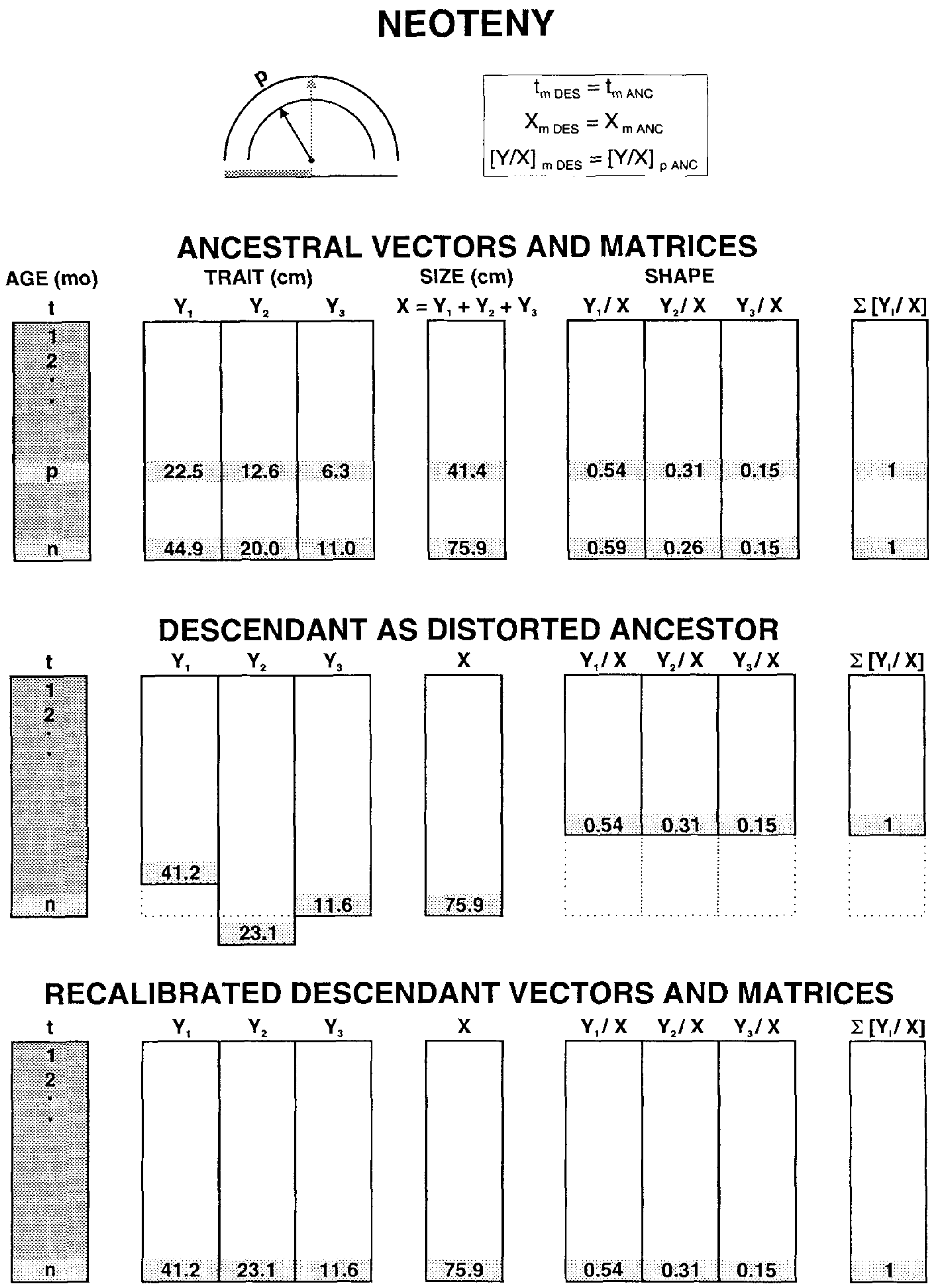


Fig. 11. Neoteny in multivariate perspective. See text.

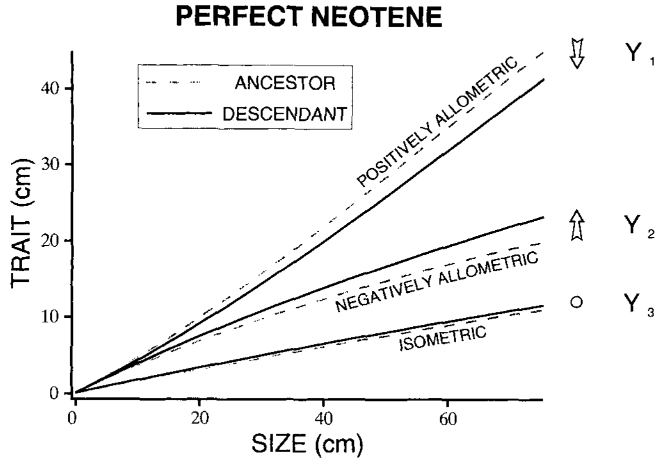


Fig. 12. Trait/size trajectories of a perfect neotene and its ancestor (as shown in Fig. 11). Despite the retardation of development relative to growth, the slopes of the descendant's trajectories are not perturbed in a uniform (downward) direction.

10 a single clock portrays every age-size-shape comparison.

Figure 11 shows that, in order for the descendant to equal the size ($X_m$) of the mature ancestor while retaining ancestral juvenile shape ($[Y_i/X]_{\mathrm{m\,DES}} = [Y_i/X]_{\mathrm{p\,ANC}}$), the growth rates of individual traits ($Y_i$) must be differentially perturbed. Trait sizes at maturation are given by the products (at maturation) of each trait's proportion of the whole, $[Y_i/X]_{\mathrm{m\,DES}}$, and overall size $X_{\mathrm{m\,DES}}$. Net growth being equal, negatively allometric traits must grow to a larger absolute size, positively allometric traits must grow to a smaller size, and isometric traits must grow to the same size in the descendant as in the ancestor. This is reflected in the varying lengths of the trait vectors depicted in the second array (i.e., the descendant vectors and matrices plotted on ancestral scales). Figure 12 shows what this implies for the slopes of descendant growth allometries: under neoteny, the slopes of trait/size relationships that are negatively allometric must be upwardly perturbed.

The reason this happens is simple. By definition, under negative allometry, the ratio of trait to overall size decreases as size increases. Let us assume no change in overall size ($\Delta X$) or time from initiation of development to maturation ($\Delta t$). In order to reduce $\Delta[Y/X]$ under these circumstances, the net growth of the trait ($\Delta Y$) must increase. An increase in $\Delta Y$ implies a smaller decrease in $Y/X$.

The inevitable conclusion is that the diagnostic tools of McKinney's "allometric heterochrony" are flawed. Reducing the rate of change in shape, $\Delta[Y/X]$, relative to growth, $\Delta X$, does not lower the slopes of growth allometries; it weakens them (Fig. 13). Negatively allometric ancestral traits grow, in neotenic descendants, with weaker negative allometry. Positively allometric ancestral traits grow, in neotenic descendants, with weaker positive allometry. Traits that grow *isometrically in ancestors remain* completely unperturbed. Under neoteny, the slopes of all growth allometries converge toward isometry. It is not the slopes of descendant growth allometries that reduce under neoteny. Rather, it is the rates of change of $Y_i/X$ that reduce.

The opposite pattern holds for acceleration. Under an increase in the rate of change of all $Y_i/X$, ancestral growth allometries strengthen. Positive allometries become more positive; negative allometries become more negative. As under neoteny, ancestral growth isometries remain unperturbed.

We conclude that Gould was correct in believing that, under a reduction of the rate of change in shape relative to growth in size, the growth rates of some traits would in-

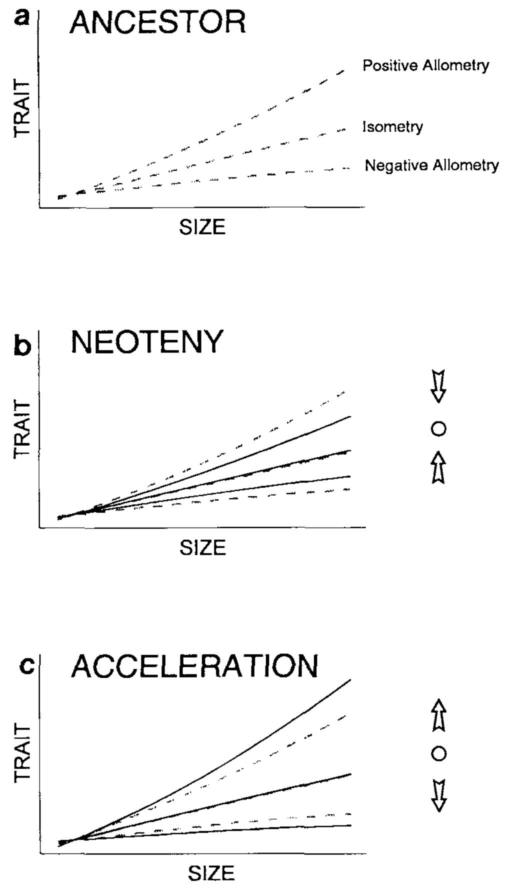


Fig. 13. Ancestral trait/size trajectories (**a**) and changes in slope induced by **b**) neoteny, and **c**) acceleration. Neoteny weakens ancestral allometries. Acceleration strengthens them. Note that the inference structure of allometric heterochrony (Fig. 2b) works only when ancestral growth allometries are positive. When ancestral growth allometries are negative or isometric, the diagnostic inference structure fails.

crease while those of others would decrease. If we were to try to model a neotene by reducing the slopes of growth allometries, we would fail. One simply cannot produce paedomorphosis of proportional shape by uniformly reducing all rates of relative growth.[8] This would require, for example, that perfect paedomorphs have relatively small heads!

The supreme irony is that this is sometimes clearly recognized in the literature on allometric heterochrony; yet it is not consistently applied. As Shea (1989:96) clearly states, under neoteny "trajectories of positive allometry require slope decreases and/or downward transpositions to yield paedomorphosis, while trajectories of negative allometry require slope increases and/or upward transpositions." Nevertheless, in the same article, Shea illustrates "true neoteny" via a decrease in a negatively allometric slope (p. 91, based on Shea, 1983, 1984).

## TWO TYPES OF DISSOCIATION

There are two distinct types of "dissociation." These are 1) the dissociation of growth fields, and 2) the dissociation of the basic processes of ontogenesis (growth, development, and maturation) that affect growth fields. Gould (1977) acknowledged the existence of the two. He called the first dissociation of primary organs. Dissociation of primary organs implies changes in rates of relative growth of different body parts. He made the second type of dissociation—dissociation of processes of ontogenesis—the focus of his treatise on ontogeny and phylogeny. Clock graphs were specifically designed to address only the latter. When evolution proceeds in a mosaic fashion, different clock graphs must be constructed for independently perturbed growth fields. The problem is that, in the literature on heterochrony, the two types of dissociation are constantly confounded.

Both types of dissociation can be diagnosed using an expanded multivariate matrix framework (Sutherland and Godfrey, in prep.). Imagine that each shape vector in the simple multivariate matrix model presented above represents a suite of trait/size proportions within a single growth field. Within growth fields, size can be conceived as the sum of the metric traits in that field, and shape as their relative proportions. Association and dissociation of growth and development can then be tested independently for each suite of traits. Patterns of association or dissociation of growth and development can be compared for different fields. Association and dissociation of growth and development across fields can be determined by calculating proportions and summing trait sizes across fields.

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[8] In fact, one cannot model an organism by reducing all rates of relative growth. If size is somehow related to the sum of the parts, and the rates of relative growth of some traits decrease, the rates of relative growth of others must increase.

In real evolutionary lineages, separate growth fields are quite commonly differentially perturbed. Ancestral linkages between the processes of ontogenesis in one field are sacrificed or preserved independently of others. Rates of growth are perturbed independently in different growth fields. When this happens, the growth fields are themselves "dissociated." However, such dissociation is not the same as dissociation of growth and development within growth fields (or within an organism as a whole).[9] Heterochronic analysis must address these as separate analytical problems (and it should not use the same vocabulary for both).

Unfortunately, in much of the literature on allometric heterochrony, "neoteny" has come to mean "dissociation" with "developmental retardation" of one growth field relative to another. This is implicit in the use of the term "neoteny," for example, to describe changes in skull/trunk relationships in chimpanzees (Shea, 1983, 1984, 1989). Pygmy chimpanzees show a downward transposition (generally through a reduction in slope) of virtually all skull/trunk allometries, whether they be negatively allometric, isometric, or positively allometric in "ancestral" common chimpanzees. But this is not "neoteny," sensu Gould. If one were to insist on modeling skull/trunk relationships in common vs. pygmy chimpanzees as a suite of heterochronic clocks, the appropriate clock would depend on the particular comparison being made.

How would Gould characterize a reduction in the slope of the relationship between log skull length ($Y$) and log trunk length ($X$) in these species? Skull length grows with negative allometry in both pygmy and common chimpanzees, but with stronger negative allometry in the former. Following Gould (1977) and Alberch et al. (1979), shape is the ratio of the metric traits under consideration (in this case, skull and trunk length) and size is the variable plotted along the abscissa. Juvenile pygmy and common chimpanzees both have large heads relative to trunk length. Adult pygmy chimpanzees, in contrast, have small heads relative to trunk length. Because both species mature at approximately the same age, the rate of change in head-to-trunk proportions must be accelerated in pygmy chimpanzees. A reduction of the slope of the skull/trunk growth allometry demonstrates peramorphosis via acceleration—not paedomorphosis via neoteny.

But the point is not that Shea is wrong in assessing pygmy chimpanzees as juvenilized in skull shape, or as retarded in the rate of skull growth (taken as a whole) relative to that of the trunk. To the contrary, he is correct. Pygmy chimpanzees are no more peramorphic than they are paedomorphic; if they were perfect peramorphs, their adult skull proportions would resemble those of overdeveloped common chimpanzees. Instead, adult skull proportions in pygmy chimpanzees are paedomorphic, just as Shea (1983) observed, but skull-to-trunk proportions are not.

The point is that a single vocabulary is being inappropriately applied to two different types of dissociation. Dissociation of growth fields has nothing to do with dissociation of growth (change in size) and development (change in shape) within a field. This conflation of two types of dissociation has resulted in species with small heads and long trunks being labeled "neotenic" even though their change in overall shape (their head-to-trunk ratio) is accelerated relative to overall growth (trunk length). Adults of these same species are labeled "paedomorphic" despite the fact that small heads on large bodies rarely characterize juveniles. What should be recognized as a reduction in a rate of relative growth is incorrectly interpreted as a reduction in a rate of change in shape relative to growth (or size). Clearly, under this new conceptualization of "dissociation" and "developmental retardation," the historical meanings of neoteny and paedomorphosis have been lost (for further analysis, see Godfrey and Sutherland, in press; Sutherland and Godfrey, in prep.).

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[9] The result of dissociation of growth fields is the production of a descendant that deviates from ancestral shape pathways (taken as a multivariate whole). The descendant is neither a paedomorph nor a peramorph; it has evolved into something new. Differential perturbations of growth and/or development in distinct growth fields are extremely common. Ancestral linkages between growth and development can be preserved in two growth fields, and yet the two can be "dissociated" because they grow and develop at different rates. We need to distinguish between dissociation of growth fields (whether this is due to changes in rates of growth and development, or to differences in linkages between of growth and development), and dissociations of growth and development within growth fields (or organisms as a whole). The two are not the same. This issue is addressed in greater detail elsewhere (Sutherland and Godfrey, in prep.).

## DEVELOLPMENTAL TIMING AND DEVELOPMENTAL RATES

What is the relationship between rates of development and the timing of events (or transitions) in a developmental sequence? Gould maintained that developmental retardation or acceleration affects the timing of developmental events. He also argued that, within a "matrix of retardation," paedomorphosis is "an almost ineluctable consequence" (1977:376). Critics have argued, contra Gould, that developmental rate has absolutely no bearing on the timing of developmental events. Furthermore, paedomorphosis is not a necessary prediction of general developmental retardation, or prolongation of life history stages.

The issue of developmental timing is important because so much of Gould's case for neoteny in human evolution depends on it, and so much of the case against Gould's hypothesis depends on the counterargument.

There are actually two separate issues at stake here. The first is the relationship between retardation of somatic development and the timing of developmental events (e.g., developmental stage transitions). The second is the relationship between developmental retardation and paedomorphosis. If somatic retardation is tied to germinal retardation in a "matrix of retardation," will the descendant adult necessarily be paedomorphic?

To test the relationship between neoteny or acceleration and the timing of developmental events (our first question), we can mark developmental events on ancestral growth trajectories, and then decrease (or increase) the rate of change in shape, tracking what happens to the timing of those particular events (Fig. 14). This can be done with or without changing the age at maturation or net growth. If Gould is correct, neoteny will delay the timing of all somatic

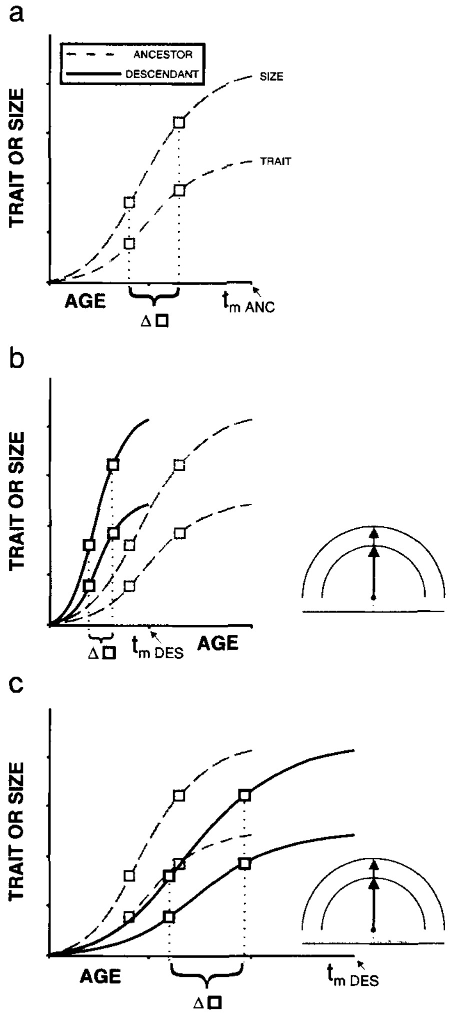

Fig. 14. Rates of development and the timing of developmental events. **a:** Ancestral size (*X*) and trait (*Y*) growth trajectories. The changing relationship between them (*Y*/*X*) represents development. Events (open squares) are embedded in the ancestral developmental sequence of the ancestor at given ages. **b:** Under rapid development, the descendant follows the ancestor's shape pathway, but achieves the ancestral mature shape, $[Y/X]_m$, at an earlier age. The growth trajectories are horizontally squeezed, as is the interval between events. **c:** Under slow development, the descendant achieves its ancestor's mature shape, $[Y/X]_m$, at a later age. The growth trajectories are horizontally stretched, as is the interval between events. Note that neither a change in overall rates of growth (which can stretch or shrink trajectories vertically) nor a change in age at maturation (which can clip or extend them) will affect the intervals between events.

developmental events, and prolong the intervals of time between them. Acceleration will hasten the timing of developmental events, and shorten the intervals between them. These changes will be independent of the age at reproductive maturation. If Gould's critics are correct, there will be no relationship between perturbations in the rate of change of shape and the timing of somatic developmental events.

Figure 14a shows ancestral growth trajectories for overall size ($X$) and trait size ($Y$). Development is their changing ratio ($Y/X$). We can model changes in the rate of development while constraining our descendant to follow ancestral developmental pathways by increasing or decreasing the amount of time it takes to accomplish the total ancestral $\Delta[Y/X]$. Thus, changing the rate of development stretches or shrinks ancestral growth trajectories horizontally. This will affect the interval between developmental events. Under rapid development, the total ancestral $\Delta[Y/X]$ is achieved in a short period of time (Fig. 14b). Under slow development, the same $\Delta[Y/X]$ takes longer to achieve (Fig. 14c).

Changing the rate of overall growth ($\Delta X$) stretches or shrinks growth trajectories vertically. If this occurs without affecting development (the proportional relationships among traits), all trait growth trajectories must be vertically distorted. Vertical distortions, however, cannot affect the intervals between developmental events.

Changing the age at reproductive maturation ($t_{\mathrm{m\,DES}}$) clips or extends growth trajectories without affecting ancestral trait/size pathways. This, too, cannot affect the intervals between developmental events. If a slowly developing descendant matures at the same age as its ancestor, the descendant's developmental trajectories will be cropped, resulting in paedomorphosis. The descendant will have traveled less along its ancestor's shape trajectory, but the long intervals between developmental events (generated by slow development) will have been preserved. If a rapidly developing descendant were to mature at the same age as its ancestor, it would pass through the ancestral suite of developmental events at a rapid pace, and then develop beyond the ancestral terminus. The descendant will have become peramorphic. However, the short intervals between ancestral developmental events will have been preserved.

The point is that the intervals between developmental events are determined solely by the horizontal stretching or shrinking of ancestral growth trajectories (affected by the rate of development)—not by vertical stretching or shrinking (affected by overall growth) or by truncation or extension (affected by age at maturation). Thus, we conclude that Gould was also correct in believing that changes in developmental rates (not growth or age at maturation) will affect the timing of somatic developmental events.

Our second question concerns the relationship between heterochronic processes and products. Is there any relationship between changes in the durations of life history stages and heterochronic products? Specifically, is Gould correct in assuming that species with prolonged life histories will also, necessarily, be paedomorphic?

Our models clearly show that, under no change in age at maturation, slow development inevitably leads to paedomorphosis in the descendant adult. However, when maturation is also retarded, the product (paedomorphosis, peramorphosis, or neither) will depend on the relative rates of somatic and germinal retardation.

Somatic development must always be more retarded than reproductive maturation in order for paedomorphosis to be manifested in the descendant adult. Paedomorphosis in descendant adults results if (and only if) development is slowed with respect to maturation. Gould (1977) seems to have understood this, because he built this requirement into his definition of neoteny and into his clock diagram of human evolution; see Figure 15a. He explicitly states that slow development does not always result in paedomorphosis (see, for example, Fig. 15b), and that peramorphosis as the expected outcome of delayed maturation: "not all types of retardation imply paedomorphosis; hypermorphosis (with recapitulation [or peramorphosis]) is also an aspect of retardation" (1977, p. 376). By his own accounting, paedomorphosis depends on development being eclipsed when still at an ancestral juvenile

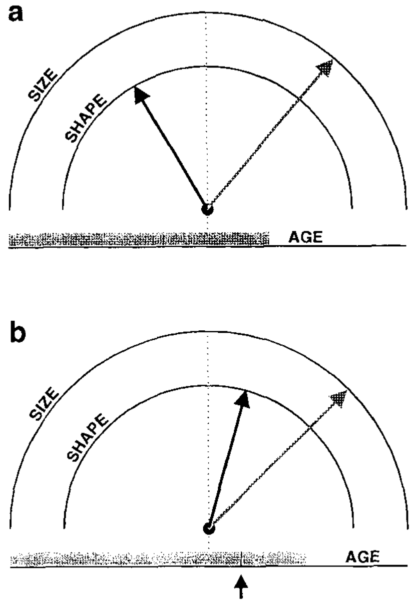

Fig. 15. **a:** Human neoteny, according to Gould (1977). **b:** Peramorphosis via slow shape transformation. Development is slowed (in time and relative to growth); nevertheless the descendant is peramorphic at maturation. Had development proceeded at ancestral rates, the descendant would have reached its mature shape at a younger age (arrow). The fact that it takes the descendant longer to reach this shape indicates that development and maturation have been dissociated.

stage; it therefore depends on the interaction between somatic development and reproductive maturation.

Neoteny, sensu Gould, involves not merely 1) retardation of development relative to growth, but also 2) retardation of development relative to maturation. In order for paedomorphosis to occur, the ancestral developmental sequence must be eclipsed. Delayed reproductive maturation decreases the likelihood of paedomorphosis in descendants, because it increases the period of time over which development occurs. This second condition for neoteny—dissociation and retardation of somatic development relative to reproductive maturation—is in many respects more important than the first.

Gould's defense of his own clock graph for human evolution is therefore less than convincing, when, in this chapter on human evolution, he uses both somatic and reproductive retardation as support for neoteny. It is here that he departs from the conditions of his own clock. Gould argues explicitly that germinal retardation alone cannot produce paedomorphosis; paedomorphosis results instead from somatic retardation. However, he fails to distinguish the opposite effects of the two when he further argues that "human retardation is a pervasive phenomenon of almost all systems, somatic and germinal. General retardation of this sort entails extensive paedomorphosis as an almost ineluctable consequence" (1977, p. 376).

Shea (1989, p. 69) found this tantamount to conflating "growth prolongation in time with morphological shape retardation." He notes, "the equation of growth prolongation with shape retardation has played a pernicious role in [the neoteny] debate, first because it is an erroneous prediction based on what we know of heterochrony, and second because the prolongation of growth periods in human evolution is so clearly true that many have simply leaped to the interpretation that the neoteny hypothesis (as so defined) must therefore also be correct" (1989:80).

Certainly, Gould (1977) is wrong when he insists that a general "matrix of retardation" must result in paedomorphosis, and Shea (1989) is correct in criticizing this aspect of Gould's argument. The irony, however, is that neither Gould nor Shea addresses the crux of the issue—i.e., the relationship between somatic and germinal retardation. Gould attributes human paedomorphosis, in essence, to somatic retardation; he uses prolongation of growth to refer to extended somatic developmental phases. Indeed, neoteny does necessitate "prolongation" of phases of somatic growth. Shea counters, in effect, that germinal retardation (prolongation in time to maturation) cannot lead to paedomorphosis. In insisting that neoteny requires prolongation of developmental stages, Gould is absolutely right. In insisting that neoteny does not require delayed maturation, Shea is absolute right. Neoteny will stand or fall as an explanation for human paedomorphosis to the extent that it can be shown that, in human ontogeny, somatic retardation exceeds germinal retardation. It is perfectly possible to test a hypothesis regarding the relationship between somatic and germinal retardation (Sutherland and Godfrey, in prep.). However, Gould's (1977: 376) simple observation that "human retardation is a pervasive phenomenon of almost all systems, somatic and germinal" does not speak to this issue at all.

Species with prolonged life histories will be no more likely to be paedomorphic than species with short life histories. Paedomorphosis (and peramorphosis) depend not on the extent to which life histories are prolonged, but on the interaction between parameters that are affected by that prolongation. The problem with Gould's analysis of human evolution is not, as critics have maintained, that slow developmental rates have nothing to do with the timing of developmental events (they most definitely do), but that a general matrix of somatic and germinal retardation does not necessarily lead to paedomorphosis. Certainly, prolongation of life history stages has as much to do with germinal as with somatic retardation. Germinal and somatic retardation have opposite effects on the production of paedomorphosis; in effect, they counter one another.

## ALLOMETRY, TIMING, AND HUMAN EVOLUTION

McKinney's "allometric heterochrony" depends strongly on three premises: first, that dissociations of growth and development generate dissociations of ancestral and descendant growth allometries; second, that changes in rates of development imply unidirectional shifts in the slopes of growth allometries; and third, that the timing of developmental events is unrelated to developmental rates. Retardation of development relative to growth is inferred whenever descendant growth allometries are deflected downward. Acceleration of development relative to growth is inferred whenever they are upwardly deflected. Association of growth and development is inferred whenever ancestral and descendant growth allometries coincide.

These premises have played a major role in critiques of Gould's neoteny hypothesis for human evolution. Some critics have denied a role for neoteny in human evolution on the grounds that human growth allometries have been perturbed in every direction, and that human ontogenesis differs from that of apes primarily in aspects of developmental timing, not rate.

Our models show that all three of these premises are wrong. Neoteny and acceleration have definite consequences for growth allometries, but they are not those assumed under the rubric of McKinney's "allometric heterochrony." Everything else (i.e., net size and age at maturation) being equal, acceleration amplifies ancestral allometries, whereas neoteny weakens them. This means that, for both acceleration and neoteny, the slopes of descendant growth allometries will be perturbed in all directions (upward, downward, and not at all, depending on the slopes of ancestral allometries). The argument that neoteny does not predict a mosaic of changes in rates of relative growth reflects a poor understanding of how slow development translates into changes in the slopes of growth allometries. Gould was correct in inferring that, under neoteny, traits growing with negative allometry (such as the brain) would grow to be larger, while traits growing with positive allometry (such as the jaws) would grow to be smaller, in the descendant than in the ancestor.

The argument that neoteny and acceleration have no bearing on timing also reflects a poor understanding of the meaning of developmental rate. Rates of development, sensu Gould (1977) and Alberch et al. (1979), are not rates of growth of traits. Developmental rates quantify changes in proportions (or relationships between structures) over time. The statement that humans do not grow slowly and are therefore not neotenic is a non sequitur. Neoteny has nothing to do with slow growth.

Confusion in the literature over the relationship between timing and rates of development has resulted in a terminological quagmire. McKinney and McNamara (1991) distinguish neoteny and acceleration from progenesis, hypermorphosis, predisplacement and postdisplacement on the grounds that the first two processes concern developmental rates, whereas the latter four concern developmental timing. To the contrary, all of these heterochronic processes affect the timing of aspects of growth and development, but in different ways. Neoteny and acceleration affect the intervals between developmental events.[10] Progenesis (paedomorphosis via early maturation) simply clips ancestral terminal events. Hypermorphosis extends development beyond the ancestral terminus, without changing the intervals between prior events. Predisplacement and postdisplacement move entire developmental sequences (earlier or later) again without changing the intervals between events.

The denial that developmental rates have anything to do with developmental timing has resulted in some rather old assessments of the role of heterochrony in human evolution. For example, in denying that neoteny and acceleration affect the timing of phases or events within developmental sequences, McKinney and McNamara (1991) have invented a new terminology to accommodate the types of changes in timing that neoteny and acceleration generate. They applied this new terminology to human evolution. There is a dominant mechanism of heterochronic change in human evolution, they state, but that mechanism is not developmental retardation. It is instead "sequential hypermorphosis" (together with "sequential postdisplacement"). In other words, humans exhibit sequentially delayed "offsets" (plus sequentially delayed "onsets") of developmental phases and events.

However, sequential hypermorphosis, along with sequential postdisplacement, is a prediction of slow development, sensu Gould. To say that humans do not develop slowly, but that we are instead sequentially hypermorphotic, is semantic obfuscation. Slow development and sequential postdisplacement-plus-hypermorphosis are one and the same.

## CONCLUSIONS

Gould's neoteny hypothesis for human evolution has been criticized on a number of grounds. The thesis of this paper is that both Gould and critics have overstated their cases. Critics contend not merely that human adults deviate in some features from the proportions and characteristics of juvenile apes (Gould admitted this), but, more importantly, that our patterns of growth—even for features that appear paedomorphic—do not match the requirements of Gould's own clock graph depicting neoteny. It is in their derivation of the expectations of Gould's clock model that, we believe, critics have erred.

According to McKinney's diagnostic formalism for allometric heterochrony, neoteny is the dissociation of ancestral and descendant growth allometries, with reduction in the slopes of the descendant allometries. Neoteny so defined is unrelated to paedomorphosis. In contrast, Gould's neoteny—the slowing of the tempo of development relative to growth in size and age at some stage of reproductive maturation (generally sexual maturation or adulthood)—has both historical precedence, and a definite logical connection to paedomorphosis.

In order to test Gould's hypothesis that humans are "essentially" neotenous apes, we must understand the expectations of Gould's neoteny model. A clear set of predictions can be formulated. For any growth field affected by neoteny, the slopes of negatively allometric traits should be higher in the descendant than in the ancestor, the slopes of positively allometric traits should be lower, and growth isometries should remain unchanged. Phases of somatic development should start later and end later in the descendant than in the ancestor—resulting in what others have called sequential postdisplacement and sequential hypermorphosis. Sexual maturation should occur when the descendant is still in an ancestral juvenile developmental stage.

Nothing that we have said should be construed as a defense of Gould's hypothesis. Our intention here is simply to reopen the dialogue, and to propose a framework for more precise testing of heterochronic hypotheses. We do believe that some aspects of Gould's hypothesis are correct—certainly human somatic development runs on a slower schedule than that of our closest relatives, and rates of overall growth in hominoids are not strongly correlated with rates of somatic development. It is not a tautology to recognize the delayed timing of developmental events as one aspect of the hypothesis of neoteny; it is a diagnostic ingredient. However, Gould's critics are also correct in asserting that slow development (with or without dissociation of growth and development) does not necessarily lead to paedomorphosis in the descendant adult. If neoteny is taken to mean paedomorphosis through a retardation of somatic development relative to 1) growth and 2) germinal maturation, then both aspects of the hypothesis must be tested.

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[10] Other heterochronic perturbations not named by Gould (1977) or Alberch et al. (1979) also affect the intervals between developmental events. These include rate hypomorphosis and rate hypermorphosis. Rate hypomorphosis lengthens the intervals, whereas rate hypermorphosis shortens them.

Gould correctly understood how rates of development affect the timing of developmental events. Rather than conflating germinal and somatic retardation, he postulated that both had occurred in the human lineage. The main problem with his analysis of heterochrony in human evolution was his failure to analyze the interaction between the two. Paedomorphosis (and peramorphosis) are not "ineluctable" consequences of retardation (and acceleration) of whole life histories.

Gould's clock graph for human neoteny (1977) is an explicit depiction of changes in the relationships between growth, development and maturation in the human evolutionary lineage. To Gould, simple dissociations of growth, development, and maturation can be invoked to explain evolutionary changes in the morphology of the human cranium and other features of human biology. To test his hypothesis for human evolution, one must understand the expectations of dissociation of the basic processes of ontogenesis. Gould's assertion that humans are "essentially" neotenic can be refuted if one can demonstrate, in field after field, that human growth allometries have been perturbed in the wrong directions. It can be refuted if somatic development in humans is nowhere "retarded" relative to both growth and age at maturation. If, however, such retardation does characterize some growth fields, then we must accept humans as at least partially, if not "essentially," neotenic.

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